

# Occupational dental erosion from exposure to acids—a review

Annette Wiegand and Thomas Attin

<b>Objective</b>	Dental erosion is characterized as a disorder with a multifactorial aetiology including environmental acid exposure. The purpose of this article was to summarize and discuss the available information concerning occupational dental erosion.
<b>Methods</b>	Information from original scientific papers, case reports and reviews with additional case reports listed in PubMed, Medline or EMBASE [search term: (dental OR enamel OR dentin) AND (erosion OR tooth wear) AND (occupational OR worker)] were included in the review. References from the identified publications were manually searched to identify additional relevant articles.
<b>Results</b>	The systematic search resulted in 59 papers, of which 42 were suitable for the present review. Seventeen papers demonstrated evidence that battery, galvanizing and associated workers exposed to sulphuric or hydrochloric acid were at higher risk of dental erosion. For other industrial workers, wine tasters and competitive swimmers, only a few clinical studies exist and these do not allow the drawing of definitive conclusions.
<b>Conclusion</b>	Occupational acid exposure might increase the risk of dental erosion. Evidence for occupational dental erosion is limited to battery and galvanizing workers, while data for other occupational groups need to be confirmed by further studies.
<b>Key words</b>	Acid; dentine; enamel; erosion; occupation; tooth wear.

## Introduction

Dental erosion is defined as the pathologic chronic loss of dental hard tissues due to the chemical influence of extrinsic and intrinsic acids without bacterial involvement [1]. The acid contact is associated with a demineralization and softening of the tooth surface, leading to an increased susceptibility to mechanical abrasion such as toothbrushing [2]. Initially, dental erosion appears as a smooth silky-shining glazed enamel surface. Further progression may lead to the development of shallow concavities or to rounding and grooving of the edges or the cusps of the tooth surfaces [3,4]. In patients with severe dental erosion, the enamel is often totally removed, leaving a vulnerable dentine surface which is often associated with a painful sensitivity and is prone to further erosion and mechanical wear. Advanced erosive tooth wear might also constitute near and frank exposures of the pulp requiring

dental treatment [5] or lead to complete destruction and tooth loss. Thus, besides preventive measures, erosive wear often requires oral rehabilitation including restorations, reconstructions or, in case of tooth loss, replacement therapies [6].

Like many oral diseases, such as dental caries, dental erosion is a disorder with a multifactorial aetiology. The main aetiological factor is the chemical dissolution of enamel and dentine by acids from exogenous or endogenous origin. During an erosive attack, protons of the acidic agent attack the components of hydroxyapatite such as carbonate, phosphate and hydroxyl ions. This attack results in dissolution of the hydroxyapatite crystals with a subsequent release of calcium ions. Of major importance for the development of dental erosions are the pH, titrable acidity, phosphate and calcium concentration and the fluoride content of the acid which determines the degree and thus the driving force of dissolution. Also, frequency and duration of acidic events have an effect on the development of erosion. However, behavioural and biological factors, such as tooth position, quality of dental hard tissues and salivary factors like composition, buffer capacity and flow rate may exert an influence on the development and progression of

Clinic for Preventive Dentistry, Periodontology and Cariology, University of Zurich, Plattenstrasse 11, CH-8032 Zurich, Switzerland.

Correspondence to: Annette Wiegand, Clinic of Preventive Dentistry, Periodontology and Cariology, University of Zurich, Plattenstrasse 11, CH-8032 Zurich, Switzerland. Tel: +41 43 6343412; e-mail: annette.wiegand@zzmk.unizh.ch

erosions. It is also suggested that presence of both acquired pellicle (bacteria-free biofilm) and microbiological plaque on tooth surfaces may impair diffusion of acids and thus the formation of erosive lesions [7–10].

Dental erosion due to intrinsic factors is caused by gastric acid reaching the oral cavity and the teeth as a result of vomiting or gastroesophageal reflux. Therefore, dental erosion is a common manifestation in patients suffering from organic or psychosomatic disorders such as anorexia or bulimia nervosa or alcohol abuse [11].

The extrinsic factors involved in dental erosion are mostly summarized under the headings diet, medications, environmental and lifestyle [12]. To date, most clinical research has focussed on the impact of acidic drinks and foods [4]. Moreover, low pH medications as well as lifestyle factors such as a lacto-vegetarian diet or drug abuse are described as risk factors for dental erosion [13–15].

Environmental acid exposure has also been associated with dental erosion and is frequently documented in case reports and several clinical surveys. However, due to the fact that environmental acid exposure has received comparatively little attention in the past, this review will concentrate on the available information concerning dental erosion induced by occupational exposure of acids.

Methods

The literature search was performed utilizing the PubMed, Medline or EMBASE database searching for the terms and their spelling variation (dental OR enamel OR dentine) AND (erosion OR tooth wear) AND (occupational OR worker) and was closely related to the MOOSE Guidelines for Meta-Analyses and Systematic Reviews of Observational Studies [16].

There was no attempt to specify the strategy in relation to data or study design. Two investigators independently screened each publication for eligibility by examining title, abstract and keywords. All original scientific papers, reviews or case reports listed in the databases were included in the review.

The following exclusion criteria were applied: (i) article does not deal with the subject, (ii) review without additional case reports dealing with occupational erosion and (iii) original papers published in languages other than English with lack of information about the prevalence of erosion in the English database abstract. References from the identified publications were manually searched to identify additional relevant articles, which were also applied to the inclusion and exclusion criteria. Data extraction was done in duplicate by both examiners. Due to the heterogeneity of type and design of the studies, further systematic analyses relating to design features to outcome of the studies were not performed. Moreover, due to differences in study design (e.g. different indices for classi-

fication of erosion and different observation periods) and the lack of existing randomized case-control studies, statistical analysis of the data seemed not appropriate for summarizing the available data.

Results

The results of the systematic search are presented in Table 1.

Most prevalence studies about occupational dental erosion were performed on workers of battery and galvanizing factories, predominately battery forming and charging workers as well as galvanizing, pickling, plating and chemical manufacturers, who are exposed to sulphuric acid and hydrochloric acid and, to a lesser degree, to phosphoric, nitric and hydrofluoric acid. One case report [17] and a total of 17 cross-sectional and non-randomized case-control studies about dental erosion published between 1961 and 2003 were obtained from the systematic search [18–34] (Table 2). For ensuring appropriate conclusions from the available data and to allow for comparison between the studies, erosions were classified into enamel and dentine erosion when decision criteria for examination were clearly defined in the publication.

From the prevalence studies including a control group (workers without occupational acid exposure), it can be summarized that the prevalence of erosion is higher in battery and galvanizing workers than in controls (Table 2). Also, aforementioned workers were more often affected from severe erosion with dentine or pulp exposure than the controls. However, it is obvious that prevalence data in both acid-exposed workers and controls exhibited a great variation amounting to 26–100% for battery and galvanizing workers and to 0–80% for

Table 1. Publications included and excluded in the review

	Number
Papers selected by the systematic search	59
Papers suitable for the review	42
Occupational groups	4
Battery and galvanizing workers and associated manufacturers	18
Industry manufacturers	6
Winemakers	12
Competitive swimmers	6
Papers excluded from the review	17
Did not deal with the topic	7
Review had no additional case report dealing with occupational erosion	2
For original papers published in languages other than English with lack of information about the prevalence of erosion in the English database abstract	8

controls [18,19,25,31–34]. Also, severe erosion with exposure of dentine varied between 14–54% in acid-exposed workers and 0–27% in controls [19,25,26,31,33,34]. In general, dental erosion induced by inhalation of acidic fumes was mostly confined to the labial and incisal surfaces of the anterior teeth [17,22,23,29].

Only five clinical studies [30,35–39] published between 1951 and 2005 and one case report [40] focussed on occupational dental erosion in workers other than battery or galvanizing manufacturers. In 1951, Elsbury *et al.* [35] examined 15 female tin factory workers who were exposed to 11 mg/m<sup>3</sup> tartaric acid dust for ~30 h a week. While none of the non-acid-exposed controls exhibited any erosion, 14 girls of the test group showed dental erosion. Early lesions could be observed after an average exposure of 10 months. Severity of erosion increased with increasing duration of employment, leading to total destruction and loss of several teeth in five workers after an average exposure time of 6 years. A small amount of data on dental erosion in munition manufacturers, soft drink manufacturers and dyestuff container cleaners was obtained from the study of ten Bruggen Cate [30]. Five of 12 munition workers and one of five soft drink manufacturers under investigation exhibited dental erosions of varying degrees. Also, enamel erosion was present in four and dentine exposure in three of seven dyestuff container cleaners [30]. Dental erosion due to the preparation of sanitary cleansers was found in five of 14 workers exposed in the process of filling domestic containers for between 3 months and 10 years [36]. ten Bruggen Cate [30] found six of 25 sanitary cleanser manufacturers to suffer from dental erosion. Only limited information could be obtained from the original paper by Goto *et al.* [41], written in Japanese. However, from 134 workers of a chemical factory in Osaka, 31% revealed signs of dental erosion [41]. The risk for occupational erosion by exposure to proteolytic enzymes or repeated exposure to acetic acid vapours by using silicone sealers was investigated by Westergaard *et al.* [38] and Johansson *et al.* [39]. Individuals working at a pharmaceutical and biotechnological enterprise showed an increased severity of facial erosion of the maxillary incisors not only with increasing exposure to proteolytic enzymes but also with age, consumption of wine and lemon tea and the use of abrasive dentifrices. Adjusted for these potential confounders, there was no association between history of occupational exposure to proteolytic enzymes and prevalent erosion [38]. For 13 subjects who had been exposed to an average of 4.2 years of working with silicone, the severity of erosion was significantly higher compared to controls. There was also a significant correlation between the period of exposure to silicone and severity of erosion [39].

The systematic search revealed four case reports [42–45], two clinical studies [46,47] and six *in vitro* studies [48–53] dealing with dental erosion due to consumption of wine. Dental erosion documented in the case reports

was related to daily average tasting of 20–30 wines over a period of 10–23 years and was predominantly located at the upper incisors [42–44]. A prevalence survey in 19 Swedish wine tasters found 78% of the subjects with dental erosion. Eleven per cent showed severe erosion with extensive exposure of dentine on multiple surfaces, 26% exhibited erosive tooth wear with localized dentine exposure and 37% showed superficial enamel erosion. The length of employment amounted to 2–37 years (median: 7 years) with the frequency of wine-tasting sessions each week varying from two to five sessions [46]. In a cross-sectional comparative study in South Africa, 21 winemakers were under investigation with a mean exposure time of 8.2 years and a number of wine tastings ranging from several tastings per week to 50–150 tastings per day [47]. Wine was kept in the mouth for 10–30 s. Only three subjects (14%) exhibited erosive tooth wear, but even so, showed a three times higher risk for dental erosion compared to non-exposed controls [47].

*In vitro* studies showed that white and red wine as well as champagne exhibit potential to cause dental erosion [48–53]. Wines contain mainly not only tartaric acid but also malic acid, lactic acid and citric acid. To a smaller amount they might also contain succinic acid, citramalic acid, galacturonic acid and mucic acid [50]. In champagne, carbonic acid is added to render sparkling. Laboratory research found that white wine (Riesling) and champagne-style wine were more erosive than red wine on both enamel and root cementum. Moreover, the erosive capability increased with increasing temperature of the respective wines [52].

Finally, two reports [54,55] and two surveys from 1983 and 1986 [56,57] indicate that competitive swimming may also be a risk factor for dental erosion. The epidemiologic survey by Centerwall *et al.* [56] reported 3% of non-swimmers, 12% of swimmers and 39% of swim team members to suffer from dental erosion. The Center for Disease Control [57] examined 30 individuals who swam five or more times a week and 60 controls and found 13% in the first group, but none of the matched controls, to suffer from enamel erosion. The assumption that dental erosion in competitive swimmers might be the result of low pH values in swimming pool waters due to an insufficient monitoring and/or inadequate buffering was analysed by Gabai *et al.* [58], who found a significant correlation between low pH, gas-chlorinated swimming pool water and general dental erosion. In contrast, Lokin and Huysmans [59] found only 0.14% of Dutch swimming pool waters, which were analysed monthly, to exhibit pH values <5.5, which is the critical pH value for enamel dissolution. Therefore, it was assumed that there is only a slight risk of swimmers developing dental erosion [55,59]. However, it is also conceived that acid drinks may be an issue as well as the pool water, as acidic drinks are likely to be consumed in large quantities by athletes [60,61].

**Table 2.** Cross-sectional and non-randomized case-control studies investigating the prevalence of erosion in battery, galvanizing and associated workers

Study	Workplace/ occupational group	Country	Number of workers under acid exposure	Number of controls	Enamel erosion	Dentine erosion	Acid type and acid concentration	Duration of acid exposure	Risk factor
Arowojolu [18]	Battery industry	Nigeria	38	67	41% <sup>a</sup>	3% <sup>a</sup>	Sulphuric acid	NA	NA
Amin <i>et al.</i> [19]	Battery industry	Jordan	24	15	6 (25%)	13 (54%)	Sulphuric acid	Mean: 11.3 years	NA
	Phosphate industry	Jordan	37	31	3 (20%) 20 (54%) 19 (61%)	4 (27%) 17 (46%) 6 (19%)	Sulphuric, phosphoric, hydrofluoric, fluosilicic acids	Mean: 9.5 years	NA
Chikte <i>et al.</i> [20]	Electroplating factory	South Africa	58	-	27 (47%)	30 (52%), tooth loss due to erosion: 13 (22.4%)	Sulphuric acid	3 months– 22 years	Five times higher risk for erosion in strippers working closest to the source than for other workers; no relationship between acid exposure time and erosion
Chikte and Josie-Perez [21]	Electro- winning facility	South Africa	103 102		22 (21%) 40 (39%)	78 (76%) 36 (35%)	Sulphuric acid, 0.3–1 mg/m <sup>3</sup> 0.1–0.3 mg/m <sup>3</sup>	1 month– 24 years Mean: 4.2 years	Five times higher risk for dentine erosion for those exposed to 0.3–1 mg/m <sup>3</sup> compared to workers exposed to 0.1–0.3 mg/m <sup>3</sup> sulphuric acid. Three times higher risk for strippers compared to other acid workers; no relationship between acid exposure time and erosion
Fukayo <i>et al.</i> [22]	Copper smelter	Japan	350	8%		Sulphuric acid		Risk for erosion increased with a history of electrolytic refining plant	
Gamble <i>et al.</i> [23]	Battery workers in five different plants	USA	245 (total) 35 (Plant A) 57 (Plant B) 38 (Plant C) 59 (Plant D) 59 (Plant E)		33 (14%) <sup>a</sup> 2 (6%) <sup>a</sup> 13 (23%) <sup>a</sup> 0 8 (14%) <sup>a</sup> 10 (17%) <sup>a</sup>		Sulphuric acid: 0.07 mg/m <sup>3</sup> 0.14 mg/m <sup>3</sup> 0.07 mg/m <sup>3</sup> 0.27 mg/m <sup>3</sup> 0.14 mg/m <sup>3</sup>	Mean: 20.2 years 4 years 10.2 years 7.5 years 12.2 years	Risk for erosion increased with increasing cumulative exposure (acid exposure × total of month worked)
Kim and Douglass [24]	34 factories (plating, galvanizing, chemical, dye and petroleum)	Korea	943		164 (17%)	78 (8%)	Various acids (e.g. hydrochloric, nitric and sulphuric acid); sulphuric acid <1 mg/m <sup>3</sup>		Risk for erosion increased with increasing exposure time

Table 2. Continued

Study	Workplace/ occupational group	Country	Number of workers under acid exposure	Number of controls	Enamel erosion	Dentine erosion	Acid type and acid concentration	Duration of acid exposure	Risk factor
Malcolm and Paul [25] and Paul [26]	Battery industry	UK	63 15	44	5% 22% 29 (46%)* 7 (47%) 0	3% 10% 26 (41%)** 0	Sulphuric acid 3.0–16.6 mg/m <sup>3</sup> 0.8–2.5 mg/m <sup>3</sup>	0–3 years >3 years 4.9–7.1 years* 12.3–16.6 years**	NA
Petersen and Gormsen [27]	Battery factory	Germany	63	31% <sup>a</sup>			Sulphuric acid 0.4–4.1 mg/cm <sup>3</sup>	16 workers: ≤10 years 47 workers >10 years	Severity of erosion increased with increasing exposure time
Remijn <i>et al.</i> [28]	Galvanizing factory	The Netherlands	38		13 (34%)	21 (55%)	Hydrochloric acid, 27% of the working time >7 mg/m <sup>3</sup>	NA	NA
Skogedal <i>et al.</i> [29]	Electrolytic zinc factory	Norway	12		1 (8%)	6 (50%)	Sulphuric acid	2–11 years	Severity of erosion increased with increasing exposure time
ten Bruggen Cate [30]	Battery factory	Great Britain	70 (formation) 16 (charging)		29 (41%) 5 (42%)	13 (19%) 0	Sulphuric acid	Up to 40 years	Severity of erosion increased with increasing exposure time
	Galvanizing factory		72 (picklers) 35 (non-picklers) 132 (other acid pickling treatments)		34 (47%) 6 (17%) 36 (27%)	7 (10%) 1 (3%) 2 (2%)	Hydrochloric, sulphuric acid Hydrochloric, nitric, sulphuric and hydrofluoric acid	Up to 40 years Up to 40 years	Severity of erosion increased with increasing exposure time Severity of erosion increased with increasing exposure time
	Plating factory		76		11 (15%)	0	Chromic, nitric, sulphuric, hydrofluoric and phosphoric acid	Up to 40 years	Severity of erosion increased with increasing exposure time
Tuominen <i>et al.</i> [31] and Tuominen and Tuominen [33,34]	Battery factories/ galvanizing factories	Finland	76	81	12% 6%	14% 5%	Sulphuric acid, 0.06–2 mg/m <sup>3</sup>	1–39 years	Prevalence of erosion increased with increasing acid exposure time: Overall: 18.4% Exposure time >13 years: 23% Exposure time >16 years: 22.4%
Tuominen <i>et al.</i> [32] Tuominen and Tuominen [34]	Fertilizer company	Tanzania	68	50	63% 38%		Sulphuric acid, 1–5 mg/m <sup>3</sup>	1–19 years	Prevalence of erosion increased with increasing acid exposure time
	Industry company	Tanzania	20	20	50% 15%		Sulphonic acid	1–19 years	

NA = information not available.

<sup>a</sup>Distribution of enamel and dentine erosion not clearly defined.

## Discussion

Our literature search has found evidence that battery, galvanizing and associated workers are at higher risk of dental erosion. Overall, the prevalence data on acid-exposed battery or galvanizing workers and controls showed great variations probably due to the multiplicity of study populations, working plants and countries. Differences in study design and the lack of existing randomized case-control studies do not allow for statistical analysis, but the available data show that the risk for dental erosion increases with increasing concentration of the acid or increasing exposure time [21,23–25,31–34] and increasing duration of employment [23–25]. Also, severity of erosion increased with increasing concentration of the acidic fumes [21,27]. With regard to the concentration of acid fumes, Chikte *et al.* [20] and Chikte and Josie-Perez [21] found a three to five times higher risk for erosion in manufacturers working closest to the acid source than for other acid workers.

Industrial workers other than battery or galvanizing manufacturers might also be at higher risk of dental erosion, but the small amount of prevalence data of chemical and pharmaceutical workers, tin and munition manufacturers and cleaners did not allow us to draw conclusions. Although the results indicate an increased risk of occupational erosion, information about the type, concentration and duration of acid exposure is lacking [30]. Moreover, the influence of possible confounders, such as medical problems of the upper respiratory tract, has to be taken into account. More prevalence data should be obtained from larger study populations to allow further appraisal of the risk of occupational dental erosion in workers.

Additionally, the small number of clinical studies in wine tasters and competitive swimmers reveals only limited data about the prevalence of dental erosion. Further information about factors contributing to the erosivity of acids or acidic fumes, such as impact of pH value, temperature and chelation potential, are required.

In view of the prevalence data of acid-exposed battery or galvanizing workers, it might be assumed that occupational dental erosion might be of higher relevance in developing compared to developed countries. Especially with regard to the prevalence studies performed after 1990, great differences between occupational dental erosion in developed and developing countries could be observed. Up to 100% of acid-exposed workers in African countries showed erosion [18–21,32,34], whereas only 8–31% of European, Korean and Japanese workers exhibited dental erosion [22,24,27,34]. Possibly, this might be a result of insufficient preventive measures to decrease acid exposure or a violation of the governmental regulations concerning maximal tolerable concentration of potentially erosive agents at workplaces. Education about occupational hazards, positive worksite oral health

promotion and training for standardized behaviours such as wearing respiratory protective equipment and gargling during/after working are considered as preventive strategies to decrease occupational erosion [24]. Also, free dental hygiene prophylactic treatment was recommended for patients having an occupation associated with an increased risk of dental erosion [62].

The reduction of the threshold limits below the level that is safe for teeth might be the measure of choice to decrease the risk of dental erosion [21,24]. The threshold limit for repeated occupational exposure to sulphuric acid or phosphoric acid in a normal 8-h workday and a 40-h workweek amounts to 1 mg/m<sup>3</sup>. The short-term exposure limit is defined as a 15-min concentration that should not be exceeded at any time during a workday and is designated to 3 mg/m<sup>3</sup> for both acids [63–66]. However, dental erosion might also be increased in workers exposed to acid concentrations below the threshold limits [21,25,27,31,33,34].

Considering erosion as a work-related condition, measures to promote occupational health are required. For individuals who are at high risk of occupational dental erosion, regular dental check-ups are recommended for the detection of early lesions and planning of preventive strategies comprising protective equipment and behaviour as well as dietary advice, optimization of fluoride regimes, stimulation of salivary flow rate, use of buffering medicaments and encouraging non-destructive toothbrushing habits [67].

## Conflicts of interest

None declared.

## References

1. Imfeld T. Dental erosion. Definition, classification and links. *Eur J Oral Sci* 1996;**104**:151–155.
2. Lussi A, Jaeggi T. Abrasion of erosion-altered dental hard tissues—a literature review. *Schweiz Monatsschr Zahnmed* 2002;**112**:629–639.
3. Lussi A, Jaeggi T, Schaffner M. Prevention and minimally invasive treatment of erosions. *Oral Health Prev Dent* 2004;**2**:321–325.
4. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. *Caries Res* 2004;**38**(Suppl. 1):34–44.
5. Sivasithamparam K, Harbrow D, Vinczer E, Young WG. Endodontic sequelae of dental erosion. *Aust Dent J* 2003;**48**:97–101.
6. Lambrechts P, Van Meerbeek B, Perdigao J, Gladys S, Braem M, Vanherle G. Restorative therapy for erosive lesions. *Eur J Oral Sci* 1996;**104**:229–240.
7. Hannig M, Balz M. Influence of in vivo formed salivary pellicle on enamel erosion. *Caries Res* 1999;**33**:372–379.

8. Johansson AK. On dental erosion and associated factors. *Swed Dent J Suppl* 2002;1-77.
9. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. *Eur J Oral Sci* 1996;104:199-206.
10. Nekrashevych Y, Hannig M, Stosser L. Assessment of enamel erosion and protective effect of salivary pellicle by surface roughness analysis and scanning electron microscopy. *Oral Health Prev Dent* 2004;2:5-11.
11. Scheutzel P. Etiology of dental erosion—extrinsic factors. *Eur J Oral Sci* 1996;104:178-190.
12. Zero DT. Etiology of dental erosion—extrinsic factors. *Eur J Oral Sci* 1996;104:162-177.
13. Duxbury AJ. Ecstasy—dental implications. *Br Dent J* 1993;175:38.
14. Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. *Scand J Dent Res* 1985;93:436-441.
15. Tredwin CJ, Scully C, Bagan-Sebastian JV. Drug-induced disorders of teeth. *J Dent Res* 2005;84:596-602.
16. Stroup DF, Berlin JA, Morton SC *et al.* Meta-analysis of observational studies in epidemiology: a proposal for reporting. *J Am Med Assoc* 2000;283:2008-2012.
17. Chikte UM, Josie-Perez AM, Cohen TL. Industrial dental erosion—a case report. *J Dent Assoc S Afr* 1996;51:647-650.
18. Arowojolu MO. Erosion of tooth enamel surfaces among battery chargers and automobile mechanics in Ibadan: a comparative study. *Afr J Med Med Sci* 2001;30:5-8.
19. Amin WM, Al Omoush SA, Hattab FN. Oral health status of workers exposed to acid fumes in phosphate and battery industries in Jordan. *Int Dent J* 2001;51:169-174.
20. Chikte UM, Josie-Perez AM, Cohen TL. A rapid epidemiological assessment of dental erosion to assist in settling an industrial dispute. *J Dent Assoc S Afr* 1998;53:7-12.
21. Chikte UM, Josie-Perez AM. Industrial dental erosion: a cross-sectional, comparative study. *SADJ* 1999;54:531-536.
22. Fukayo S, Nonaka K, Shinozaki T, Motohashi M, Yano T. [Prevalence of dental erosion caused by sulfuric acid fumes in a smelter in Japan]. *Sangyo Eiseigaku Zasshi* 1999;41:88-94 [in Japanese].
23. Gamble J, Jones W, Hancock J, Meckstroth RL. Epidemiological-environmental study of lead acid battery workers. III. Chronic effects of sulfuric acid on the respiratory system and teeth. *Environ Res* 1984;35:30-52.
24. Kim HD, Douglass CW. Associations between occupational health behaviors and occupational dental erosion. *J Public Health Dent* 2003;63:244-249.
25. Malcolm D, Paul E. Erosion of the teeth due to sulphuric acid in the battery industry. *Br J Ind Med* 1961;18:63-69.
26. Paul E. Erosion of the teeth due to industrial sulphuric acid. *Dent Mag Oral Top* 1961;79:137-143.
27. Petersen PE, Gormsen C. Oral conditions among German battery factory workers. *Community Dent Oral Epidemiol* 1991;19:104-106.
28. Remijn B, Koster P, Houthuijs D *et al.* Zinc chloride, zinc oxide, hydrochloric acid exposure and dental erosion in a zinc galvanizing plant in the Netherlands. *Ann Occup Hyg* 1982;25:299-307.
29. Skogedal O, Silness J, Tangerud T, Laegreid O, Gilhuus-Moe O. Pilot study on dental erosion in a Norwegian electrolytic zinc factory. *Community Dent Oral Epidemiol* 1977;5:248-251.
30. ten Bruggen Cate HJ. Dental erosion in industry. *Br J Ind Med* 1968;25:249-266.
31. Tuominen M, Tuominen R, Ranta K, Ranta H. Association between acid fumes in the work environment and dental erosion. *Scand J Work Environ Health* 1989;15:335-338.
32. Tuominen ML, Tuominen RJ, Fubusa F, Mgalula N. Tooth surface loss and exposure to organic and inorganic acid fumes in workplace air. *Community Dent Oral Epidemiol* 1991;19:217-220.
33. Tuominen M, Tuominen R. Dental erosion and associated factors among factory workers exposed to inorganic acid fumes. *Proc Finn Dent Soc* 1991;87:359-364.
34. Tuominen M, Tuominen R. Tooth surface loss and associated factors among factory workers in Finland and Tanzania. *Community Dent Health* 1992;9:143-150.
35. Elsbury WB, Browne RC, Boyes J. Erosion of teeth due to tartaric acid dust. *Br J Ind Med* 1951;8:179-180.
36. Lapping D. Dental erosion in a South African chemical industry. *S Afr Med J* 1964;38:15-16.
37. Goto H, Kosaka M, Ueda T, Yoshida M, Hara I. Association between dental erosion and exposure to acids in a chemical factory. *Sangyo Eiseigaku Zasshi* 1996;38:165-171.
38. Westergaard J, Larsen IB, Holmen L *et al.* Occupational exposure to airborne proteolytic enzymes and lifestyle risk factors for dental erosion—a cross-sectional study. *Occup Med (Lond)* 2001;51:189-197.
39. Johansson AK, Johansson A, Stan V, Ohlson CG. Silicone sealers, acetic acid vapours and dental erosion: a work-related risk? *Swed Dent J* 2005;29:61-69.
40. Westergaard J, Moe D, Pallesen U, Holmen L. Exaggerated abrasion/erosion of human dental enamel surfaces: a case report. *Scand J Dent Res* 1993;101:265-269.
41. Goto H, Kosaka M, Ueda T, Yoshida M, Hara I. Association between dental erosion and exposure to acids in a chemical factory. *Sangyo Eiseigaku Zasshi* 1996;38:165-171.
42. Gray A, Ferguson MM, Wall JG. Wine tasting and dental erosion. Case report. *Aust Dent J* 1998;43:32-34.
43. Chaudhry SI, Harris JL, Challacombe SJ. Dental erosion in a wine merchant: an occupational hazard? *Br Dent J* 1997;182:226-228.
44. Ferguson MM, Dunbar RJ, Smith JA, Wall JG. Enamel erosion related to winemaking. *Occup Med (Lond)* 1996;46:159-162.
45. Mandel L. Dental erosion due to wine consumption. *J Am Dent Assoc* 2005;136:71-75.
46. Wiktorsson AM, Zimmerman M, Angmar-Mansson B. Erosive tooth wear: prevalence and severity in Swedish winetasters. *Eur J Oral Sci* 1997;105:544-550.
47. Chikte UM, Naidoo S, Kolze TJ, Grobler SR. Patterns of tooth surface loss among winemakers. *SADJ* 2005;60:370-374.
48. Chikte UM, Grobler SR, Kotze TJ. In vitro human dental enamel erosion by three different wine samples. *SADJ* 2003;58:360-362.
49. Lupi-Pegurier L, Muller M, Leforestier E, Bertrand MF, Bolla M. In vitro action of Bordeaux red wine on the microhardness of human dental enamel. *Arch Oral Biol* 2003;48:141-145.

50. Rees J, Hughes J, Innes C. An in vitro assessment of the erosive potential of some white wines. *Eur J Prosthodont Restor Dent* 2002;**10**:37–42.
51. Lussi A, Jaggi T, Scharer S. The influence of different factors on in vitro enamel erosion. *Caries Res* 1993;**27**:387–393.
52. Mok TB, McIntyre J, Hunt D. Dental erosion: in vitro model of wine assessor's erosion. *Aust Dent J* 2001;**46**:263–268.
53. Willershausen B, Schulz-Dobrick B. In vitro study on dental erosion provoked by various beverages using electron probe microanalysis. *Eur J Med Res* 2004;**9**:432–438.
54. Geurtsen W. Rapid general dental erosion by gas-chlorinated swimming pool water. Review of the literature and case report. *Am J Dent* 2000;**13**:291–293.
55. Scheper WA, van Nieuw AA, Eijkman MA. Oral conditions in swimmers. *Ned Tijdschr Tandheelkd* 2005;**112**:147–148.
56. Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. *Am J Epidemiol* 1986;**123**:641–647.
57. Leads from the MMWR. Erosion of dental enamel among competitive swimmers—Virginia. *J Am Med Assoc* 1983;**250**:716.
58. Gabai Y, Fattal B, Rahamin E, Gedalia I. Effect of pH levels in swimming pools on enamel of human teeth. *Am J Dent* 1988;**1**:241–243.
59. Lokin PA, Huysmans MC. Is Dutch swimming pool water erosive? *Ned Tijdschr Tandheelkd* 2004;**111**:14–16.
60. Milosevic A, Kelly MJ, McLean AN. Sports supplement drinks and dental health in competitive swimmers and cyclists. *Br Dent J* 1997;**182**:303–308.
61. Sirimaharaj V, Brearley Messer L, Morgan MV. Acidic diet and dental erosion among athletes. *Aust Dent J* 2002;**47**:228–236.
62. Lussi A, Jaeggi T. Occupation and sports. *Monogr Oral Sci* 2006;**20**:106–111.
63. American Conference of Governmental Industrial Hygienists. Hydrogen chloride: TLV Chemical Substances 7th edition Documentation 2003; Publication #7DOC-311.
64. Japan Society for Occupational Health. Recommendation of occupational exposure limits. *J Occup Health* 2004;**46**:329–344.
65. American Conference of Governmental Industrial Hygienists. Sulfuric acid: TLV Chemical Substances 7th Edition Documentation 2004; Publication #7DOC-555.
66. National Institute for Occupational Safety and Health. *NIOSH Pocket Guide to Chemical Hazards*. Washington DC: NIOSH, Publication No. 2005–151, 2005.
67. Lussi A, Hellwig E. Risk assessment and preventive measures. *Monogr Oral Sci* 2006;**20**:190–199.